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SCOGS report on the health aspects of sucrose consumption

Dear Sir:

We would like to voice our disappointment over the report on the health aspects of sucrose consumption issued by the Select Committee on GRAS substances (1). It is concluded in this report that except for dental caries, there is no clear association between the present level of sucrose consumption and the incidence of vascular degenerative disease or diabetes in this country.

As we indicated in the material submitted to the public hearing held by the FDA, there is abundant evidence showing that dietary sucrose is one of the dietary factors responsible for obesity, diabetes and heart disease in this country. While a detailed repetition of the evidence is beyond the scope of this letter, we would like to discuss the points raised by the report.

The report states that "Findings linking ingestion of sucrose with diabetes are essentially circumstantial. There is no plausible evidence that sucrose, except as it is a nonspecific source of excessive calories, is related to the disease." That statement might have been true in the past. At present, there are numerous studies which show that sucrose (as compared to starch) increases fasting serum insulin (2-5) and decreases the insulin sensitivity of adipose tissue (4-6). There is also ample evidence to show that in the process of acculturation to higher living standards there is a dramatic rise in the number of cases of diabetes two decades after acculturation begins (7-9), and that the rise in the rate of diabetes is closely followed by a rise in the occurrence of vascular disease (7). It may be said that the role of sucrose in these processes cannot be factored out, since acculturation involves a rise in both the intake of sugar and the intake of saturated fat and cholesterol. However, the dramatic and tragic rise in diabetes among Yemenite Jews in the last 30 years has occurred without a significant rise in saturated fat intake and is traceable to one and only one dietary factor: a marked increase in the consumption of sucrose by

these people since they entered Israel (9, 10).

The Committee report emphasizes that high levels of sucrose must be consumed in order to demonstrate undesirable metabolic changes. The premise, that high levels of anything will produce adverse effects and that, therefore, these adverse effects do not reflect a true dietary hazard, is false. Conceding that in most studies sucrose is fed at a level higher than that consumed by the average American, the report ignores four important points:

- 1) Some individuals consume much more sucrose than the average intake.
- 2) Studies with sucrose are comparative; that is, the effects of a large amount of sucrose are compared to the effects of an equal amount of starch; and while sucrose can be shown to produce adverse effects including all the symptoms of diabetes in susceptible animals (11-14), the same level of starch in the same type of animal does not produce any symptoms of diabetes (2).
- 3) In animal studies, virtually all the clinical symptoms of diabetes can be produced by feeding high levels of sucrose (11-14), but these symptoms are not produced by feeding high levels of dietary fat or cholesterol.
- 4) Metabolic changes observed in homogeneous animal species fed high levels of sucrose would be expected to occur in a finite portion of a heterogeneous human population consuming lower levels of sucrose.

The report acknowledges that a segment of the population appears to have a genetic predisposition to experience a large and permanent increase in blood triglycerides when consuming diets containing sucrose. In sensitive individuals, these effects have been observed with sucrose levels as low as 20-25% of the total caloric intake (15)—well within the range of the average sucrose intake. This type of hyperlipidemia has been associated with abnormal glucose tolerance, diabetes and heart disease. It is difficult to



see how sucrose intake at the present level represents no health hazard to the approximately 15 million carbohydrate sensitive adults in this country.

In the report the point is made that sucrose may act synergistically with dietary fat to increase the level of blood lipids. The report considers the role of sucrose as secondary to that of lipids and states that cholesterol must be added to a sucrose diet in order to produce hypercholesterolemia. This implies that dietary cholesterol and sucrose constitute an unusual dietary combination and that, therefore, the results are of little importance. In fact, the average American diet contributes 600 mg cholesterol and 150 g sucrose/day per person. Considering that the bulk of blood cholesterol is synthesized in the liver from carbohydrate, the cholesterol-sucrose synergism on the blood picture is of great importance. Similarly, the synergistic action of saturated fat and sucrose in increasing blood triglyceride levels makes sucrose an important hypertriglyceridemic agent, since saturated fat composes 16% of the total calorie intake in the average American diet. The findings not stressed in the report are that the isocaloric replacement of sucrose by glucose or glucose polymers prevents these synergistic increases in blood lipids.

In conclusion, we believe that reasonable scientific evidence exists to conclude that:

- 1) Sucrose alone may be a very important etiological factor in heart disease and diabetes in that segment of the population described as carbohydrate sensitive (10% of the population).
- 2) In most of the population (90%), sucrose may not be a primary risk factor by itself, but by virtue of its synergistic interaction with dietary cholesterol and triglyceride, sucrose still must be considered an important risk factor in the development of vascular disease and diabetes.

In view of the foregoing, we strongly recommend that sucrose intake from all sources except fresh or processed fruit (without added sugar) be decreased by a minimum of 60% and be replaced by complex carbohydrate from foods such as vegetables and cereals. The implementation of this recommendation will entail that the

sucrose content of packaged, frozen and canned foods be determined and displayed and that a national campaign be launched to inform the populace of the hazards of excessive sugar consumption. In addition, a concerted national effort should be made to identify carbohydrate-sensitive individuals. Sucrose intake in these individuals should be exclusively from fresh or processed fruit (without added sugar).

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References

- SCOGS-69. Evaluation of the health aspects of sucrose as a food ingredient. Contract No. FDA 223-75-2004. Life Sciences Research Office, FA-SEB, 1976.
- COHEN, A. M., A. TEITELBAUM, S. BRILLER, L. YANKO, E. ROSENMANN AND E. SHAFRIR. Experimental models of diabetes. In: Sugars in Nutrition, edited by H. Sipple and K. McNutt. New York: Academic Press, 1974, p. 483.
- SZANTO, S. AND J. YUDKIN. The effect of dietary sucrose on blood lipids, serum insulin, platelet adhesiveness and body weight in human volunteers. Postgrad. Med. J. 45: 602, 1969.
- 4. BLAZQUES, E., AND C. L. QUIJADA. The effect of a high-carbohydrate diet on glucose, insulin sensitivity and plasma insulin in rats. J. Endocrinol. 44: 107, 1969.
- REISER, S., AND J. HALLFRISCH. Insulin sensitivity and adipose tissue weight of rats fed starch or sucrose diets ad libitum or in meals. J. Nutr. 107: 147, 1977.
- VRÁNA, A., Z. SLABOCHOVÁ, L. KAZDOVÁ AND P. FÁBRY. Insulin sensitivity of adipose tissue and serum insulin concentration in rats fed sucrose or starch diets. Nutr. Rept. Internatl. 3: 31, 1971.
- CLEAVE, T. L., G. C. CAMPBELL AND W. S. PAINTER. Diabetes, Coronary Thrombosis and the Saccharide Disease, (2nd ed.). Bristol: John Wright and Sons, 1969.
- CAMPBELL, G. D. Diabetes in Asians and Africans in and around Durban. S. Afr. Med. J. 37: 1195, 1963.
- COHEN, A. M. Prevalence of diabetes among different ethnic Jewish groups in Israel. Metabolism 10: 50, 1961.
- COHEN, A. M. Fats and carbohydrates in atherosclerosis and diabetes in Yemenite Jews. Am. Heart J. 65: 291, 1963.
- 11. COHEN, A. M., A. TEITELBAUM AND R. SALITER-NIK. Genetics and diet as factors in development



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of diabetes mellitus. Metabolism 21: 235, 1972.

12. Cohen, A. M., and E. Rosenmann. Diffuse glomerulosclerosis in sucrose-fed rats. Diabetolo-

gia 7: 25, 1971.

- ROSENMANN, E., A. TEITELBAUM AND A. M. COHEN. Nephropathy in sucrose-fed rats. Electron and light microscopic studies. Diabetes 20: 803, 1971.
- 14. Cohen, A. M., I. C. Michaelson and L.
- YANKO. Retinopathy in rats with disturbed carbohydrate metabolism following a high sucrose diet.

 I. Vascular changes. Am. J. Ophthol. 73: 863, 1972.
- 15. Nikkila, E. A. Influence of dietary fructose and sucrose on serum triglycerides in hypertriglyceridemia and diabetes. In: Sugars in Nutrition, edited by H. Sipple and K. McNutt. New York: Academic Press, 1974, p. 439.